squares (□) indicate the results of MCA-occluded rats administered with ginsenoside Rb_1 in a dose of 60 μg/day.

As shown in Fig. 1, the place navigation disability after MCA permanent occlusion (after cerebral infarction) was significantly improved by ginsenoside Rb_1 infusion in groups of cerebral infarction administered with ginsenoside Rb_1 as compared with a group of cerebral infarction administered with physiological saline. Especially, in the water maze tests at the 2nd week and at the 4th week after MCA occlusion, the low dose of ginsenoside Rb_1 significantly ameliorated the learning disability on the 3rd day and on the 4th day, and the high dose of ginsenoside Rb_1 on the 4th day at the 2nd week and on the 3rd and 4th days at the 4th week after MCA occlusion. Significant effects were also noted on the 1st day at the 4th week in the high dose and the low dose groups, respectively. No significant differences in swimming speed of SH-SP rats were detected among the four experimental groups.

After the water maze tests at the 4th week, the SH-SP rats were anesthetized with chloral hydrate, and they were perfused and fixed transcardially with 0.1 mole phosphate buffer containing 4% paraformaldehyde. The brains were dissected out and cerebrocortical infarcted areas were photographed. Areas of the left cerebral hemispheres and the left cerebrocortical infarct lesions were measured on the photographs by using an image analysis device. The left cerebrocortical infarcted areas

were divided by the left cerebral hemispheric areas to calculate ratios of the cerebrocortical infarction (%). Results are shown in Fig. 2.

As shown in Fig. 2, the ratio of cerebrocortical infarction was significantly reduced in the groups of cerebral infarction with intravenous administration of ginsenoside Rb_1 as compared with the group of cerebral infarction with administration of physiological saline. Since the ratio of cerebrocortical infarction is calculated based on the area of infarction, and the mean value of the ratio in the groups intravenously administered with ginsenoside Rb_1 is reduced to about 50% or less compared with that of the group administered with physiological saline, actual volume of infarction appears to be reduced to about 1/4 by intravenous administration of ginsenoside Rb_1.

An actual case of cerebral infarct area of the group administered with physiological saline and an actual case of cerebral infarct area of the group administered with ginsenoside Rb_1 (6μg/day) are shown in Fig. 3A and Fig. 3B, respectively.

Fig. 4 is a schematic drawing summarizing the results of the present experiments. In rats administered with physiological saline, the size of cerebral infarction remained large and it took a long time for the rats to escape onto the goal platform in the water maze tests. Contrary, in rats administered with ginsenoside Rb_1 of the present invention, the infarct area was reduced, and as a result, in the water maze

tests, only a short time was required for the rats to arrive at the goal platform.

According to the paper in the past by the inventor (Sakanaka) using the transient forebrain ischemia model of gerbils (Wen T.-C., et al., Acta Neuropathol., 91, 15-22, 1996), even if intraperitoneal administration of ginsenoside Rb_1 (10 mg/kg/day or 20 mg/kg/day) was performed before ischemic loading, only about 30% of hippocampal CA1 pyramidal neurons could be rescued. In addition, intraperitoneal administration of ginsenoside Rb_1 in gerbils after the ischemic insult resulted in no effect. Moreover, since the daily doses of intraperitoneally administered ginsenoside Rb_1 are as high as 0.7 mg - 1.4 mg determined by the body weight of gerbils (approximately 70 g), judging from the view point of efficacy and effect of ginsenoside Rb_1 administration, intravenous administration of ginsenoside Rb_1 is a superior method for administration than the intraperitoneal administration, and can be easily applied to humans. As well known, an intraperitoneal administration to human can not always be applied except for a partial exception (peritoneal lavage, etc.).

Animals with MCA permanent occlusion (cerebral infarction rats or cerebral embolism rats) used in the present example are obviously more severe than the transient forebrain ischemia model of gerbils and they provide a model close to human disease that is cerebral infarction. Consequently, the fact that the